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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER
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LI, QIAN J

ART UNIT	PAPER NUMBER
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1632

DATE MAILED: 11/19/2001

13

Please find below and/or attached an Office communication concerning this application or proceeding.

# Office Action Summary

Application No.

09/511,008

Applicant(s)

HAGEMAN, GREGORY S.

Examiner

Janice Li

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

## Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

## Status

- 1) ☐ Responsive to communication(s) filed on \_\_\_\_.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

## Disposition of Claims

- 4) ☒ Claim(s) 1-37 is/are pending in the application.
- 4a) Of the above claim(s) 8, 11, 13-18, 20-35 and 38-66 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-7, 9, 10, 12, 19, 36, 37 and 67 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_ are subject to restriction and/or election requirement.

## Application Papers

- 9) ☒ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on \_\_\_\_ is: a) ☐ approved b) ☐ disapproved by the Examiner.
- If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

## Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
- a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

## Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) g.
- 4) ☐ Interview Summary (PTO-413) Paper No(s). \_\_\_\_.
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: \_\_\_\_\_.

## DETAILED ACTION

### *Election/Restrictions*

Applicant's provisional election with traverse of Group II, and species of elastin, in Paper No. 12 is acknowledged. The traversal is on the ground(s) that Group II and III are classified in the same class, and that the polynucleotide markers recited in Group III include those encode protein markers. Regarding election of species, applicants provide choice of the species of drussen-associated markers or elastin and further argue that many markers recited in claim 10 would be obvious over each other, such as different immunoglobulin chains. In response, the arguments are not found persuasive because

- 1). Groups II and III are classified in different subclasses, thus are considered as different classification. Groups II and III are drawn to different methods detecting patentably distinct molecules, such as immunochemistry and genotyping.
- 2). Although the polynucleotide recited in Group III include those encoding protein markers of group II, they further encompass much more nucleic acids, such as gene mutation, deletion, and genome polymorphism taught in the specification.
- 3). The drussen-associated markers recited in claims 9 and 10 are not limited to immunoglobulin chains, they embrace many pathological processes which might be the cause or secondary effects of drussen formation, and encompass various immunoglobulins, complements, cellular membrane receptors, extracellular matrix components, cytokines, adhesion molecules and more. A serious search burden would be imposed to the Office if groups II, III, and all drussen-associated markers are examined together.

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Applicants further request to traverse the restrictions to groups VIII-X, this is irrelevant to the current prosecution, and the request would be considered at the time a related divisional application is filed.

Therefore, it is maintained that each of the Inventions II and III requires a separate search status and consideration. The inventions are mutually exclusive and independent methods for detecting protein markers and gene markers. The different methods have different method steps, modes of operation, and criteria of measurement, use patentably distinct reagents (nucleic acid primers and probes, and antibodies, etc.), and have distinct technical considerations. Therefore, it is maintained that these inventions are distinct due to their divergent subject matter. Further search of these inventions is not co-extensive, as indicated by the separate classifications. The requirement is still deemed proper and is therefore made **FINAL**.

However, please note that as per Applicants' request claim 21 will be rejoined and examined to the extent that reads on the elected invention.

Please note that after a final requirement for restriction, the Applicants, in addition to making any response due on the remainder of the action, may petition the Commissioner to review the requirement. Petition may be deferred until after final action on or allowance of claims to the invention elected, but must be filed not later than appeal. A petition will not be considered if reconsideration of the requirement was not requested. (See § 1.181.).

Claims 1-67 are pending, however, claims 8, 11, 13-18, 20-35, and 38-66 are withdrawn from further consideration by the Examiner, pursuant to 37 CFR 1.142(b), as

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being drawn to non-elected inventions, there being no allowable generic or linking claim.

Claims 1-7, 9, 10, 12, 19, 36, 37, and 67 are under current examination.

### ***Specification***

The specification is objected to because of the following informalities: pages of the specification should be numbered continuously in Arabic numbers, numbering pages as 94, 94A, 94B or 98, 98A, and 98B, for example, are not legible for publication, appropriate correction to all such practice is required.

### ***Claim Rejections - 35 USC § 112***

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-7, 9, 10, 12, 19, 36, 37, and 67 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Claims are drawn to a method and a kit for diagnosing or determining a predisposition to developing an arterial wall disruptive disorder in a subject comprising detecting markers, such as elastin, for macular degeneration in the eye, wherein said marker is indicative of an arterial wall disruptive disorder.

The nature and breadth of the claims pertain to a diagnostic method for prediction and diagnosis of a vascular disruptive disorders, particularly aneurysm, by means of various findings associated with macular degeneration in the eye, particularly AMD. These markers include genes and proteins associated with a wide variety of biochemical and physiological processes such as immunological events (dendritic cells, autoantibodies, cytokines, HLA markers, complements), extracellular matrix (ECM) metabolic pathways, lipid metabolic pathways, biogenesis of fibrosis, and blood clotting events, etc.

There are many factors to be considered when determining whether the disclosure satisfies the enablement requirements and whether undue experimentation would be required to make and use the claimed invention (see *In re Wands*, 858 F. 2d 731, 737, 8 USPQ 2d 1400, 1404, 1988). These factors include but are not limited to the nature of the invention, the state of the prior art, the relative skill of those in the art, the predictability of the art, the breadth of the claims, and amount of direction provided.

An enabled diagnostic method should be able to provide a requisite standard or an aid for the determining of the nature of a disease or for the distinguishing of one disease from another. In the instant case, to identify AAA assessed by a protein marker such as elastin.

In view of the guidance provided, the specification teaches that there are similarities in general characteristics of various aneurysm and AMD, such as heritable, age-related, collagen and elastin neosynthesis, smoking as a risk factor, influx of dendritic cells, and upregulation of MMPs (pages 20, 65-67); that some of the ECM

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changes occurred in both AMD and AAA (table 1, page 48 and tables B&C, page 98B); that of 33 donors with AMD, eight also had AAA (24.2% of AMD donors, tables 2 and 3, pages 91-93). However, the specification fails to teach whether such co-incidence is significant over other influential risk factors, such as aging, how the trends, degrees, and anatomic location of changes of these markers are related to the onset or development of arterial wall disruptive disorders. For example, when a patient has an elevated drusen elastin level, how to make the diagnosis for AAA? One possible way to address the question would be finding the prevalence of AAA in a population having abnormal elastin levels. The specification fails to provide such information, thus, it fails to provide an enabling disclosure to support the claims.

In considering a valid marker for diagnosis of a vascular disruptive disorder, it is noted that prior art acknowledges that aortic aneurysm and macular degeneration are belong to the same disease category in that they both are modulated by metalloproteases, which are important in extracellular matrix remodeling (US 6,197,770, claims 38, 39, column 23, lines 57-63; column 26, lines 14-26), that arterial wall disorders may be involved in the etiology of AMD (*Vingerling et al*, *Am J Epidemiol* 1995;42:404-9). Thus, it is known in the art that one could potentially be a risk factor for the other. However, it is not known what would be the difference between the two disease categories, how the risk factors and descriptive markers be used as a diagnostic tool. It is also noted that the etiology for AAA is still unclear and most likely associated with complex interactions among hereditary factors, environmental factors, and aging process, most of the studies published are observatory (*Boyle et al* *IDS*,

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*Bilato et al* IDS, *Cattell et al* IDS); "ANEURYSMAL DISEASE IS CHARACTERIZED BY A PROGRESSIVE DEPLETION OF ELASTIN" (*Boyle et al*), and the evaluation to elastin levels in macular degeneration is rarely seen on record, and characterized by increased levels of elastin according to the specification. Thus, the state of the art in general has yet to determine whether AAA and AMD are the same in nature, whether and how the markers for AMD could be used for diagnosis of AAA, and the relative accuracy of such practice. Therefore, it is incumbent for applicants to provide adequate disclosure to guide the practice of the invention within the specification. However, the specification fails to teach, either from a population study or from a case-control study, the elastin levels in the serum and/or the drusen in normal control, in AAA population and AMD population, the prevalence of both diseases in the population having abnormal elastin levels; the specification fails to provide particulars regarding how elastin would change in localized lesion, and in the serum of AMD, or the co-incidence of AAA and AMD in a larger disease population, it fails to provide an established relationship between AMD and AAA, and among the levels of elastin or other recited protein markers, and AMD or AAA. Therefore, the results would be highly unpredictable if one skilled artisan uses AMD markers to diagnose vascular disruptive disorders.

The following are some further noted barriers for practice the claimed invention.

With regard to working example 1, the specification teaches that of 33 AMD patients, 8 also has AAA, thus, if applying this probability to predict AAA, 75% of AMD patients would not have AAA, thus, generally applying AMD markers for diagnosis of AAA seems questionable. Further, the assessment is conducted in a very small number



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(33) of AMD cases in a retrospective study, it is not necessarily representative to the entire population of AMD or AAA. Thus, in a highly unpredictable art, 33 cases are too small a sample to represent the AAA prevalence in the AMD population.

In assessing the value of ECM components and vascular diseases, *Robert et al* (Atherosclerosis 1998;140:281-295, IDS-BV) teach evaluating atherosclerosis (Thickening and loss of elasticity of arterial walls) by levels of elastin peptides, elastase activity, and elastase inhibitory activity, which illustrates the complicity of elastin metabolism. "IN ORDER TO RENDER ACCESSIBLE VASCULAR MATRIX MODIFICATIONS TO CLINICAL STUDIES, EXPERIMENTS HAD TO BE UNDERTAKEN ON AN EPIDEMIOLOGICAL LEVEL TO CORRELATE THE WIDELY ACCEPTED CLINICAL PARAMETERS OF ATHEROGENESIS WITH DETERMINATIONS OF MATRIX METABOLITES IN THE SAME BLOOD SAMPLES. THE MAIN DIFFICULTY WAS TO DECIDE WHICH BLOOD PARAMETERS MIGHT BE RELATED TO VASCULAR MATRIX MODIFICATIONS?", "WITHOUT SUCH MODIFICATIONS, ECM PARAMETERS WOULD NOT BECOME ACCESSIBLE FOR CLINICAL EVALUATION". The instant specification fails to teach the elastin levels in individuals and groups of individuals having AMD+/AAA+ and AMD-/AAA+ to establish a relationship between markers and diseases.

With regard to the levels of elastin in AAA, *Grange et al* (Cardiovascular Surg 1997;5:256-65, IDS-AZ) teach "ONE OF THE MOST STRIKING HISTOLOGIC FEATURES OF ANEURYSMAL TISSUE IS THE FRAGMENTATION OF THE MEDIAL LAMELLAE AND DECREASED CONCENTRATION OF ELASTIN". They also teach that even though no evidence for synthesis of new elastin fibers beyond the perinatal period, the mass of elastin in the adult thoracic aorta to be twice as much as that of a child, and low levels of elastin mRNA is detectable in adult aorta in some study (left column, page 259). Thus, elastin levels

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apparently vary among different age groups, and decreased histologically in aneurysmal tissue. *Sobolewski et al* (IDS-BZ) teach the amount of elastin in aneurysms is significantly lower, whereas the quantitative ratio between collagens of various types does not differ significantly, only solubility of the collagen and its susceptibility to the action of EDTA are distinctly decreased. As for the levels of elastin in macular degeneration, although there are many descriptive studies of ECM components, record is silent regarding the elastin levels in macular degeneration or in drusen. The instant specification teaches in tables 1 and D that expression of elastin and its mRNA in choroidal fibrosis are both "increased", which is **contrary**, at least at protein levels, to the histological findings in aneurysmal tissue as taught by *Grange et al* and *Sobolewski et al*. The specification fails to teach, either from population study or from individual study, the elastin levels in normal control, in AAA population and in AMD population, how the changes of elastin associated with both diseases, a simple notion of "increased" in the table is not sufficient to enable the practice of the invention, i.e. using elastin changes for diagnosis and prediction of macular degeneration. Therefore, from the teachings of the specification, one skilled in the art could not determine whether the decrease or increase of elastin is indicative of AAA, in what degree such prediction is accurate, one skilled artisan could not practice the invention without extensive undue experimentation.

With regard to assessing elastin levels, elastin is present as part of the normal extracellular matrix homeostasis. *Robert et al* (IDS-BZ) teach measuring a combination of levels of elastin peptides, elastase activity, and elastase inhibitory activity, which

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illustrates the complicity of elastin metabolism, and the presence of a constant equilibrium among the elastase and inhibitory forces, as such, simple measurement of elastin seems not as straight forward in diagnosis of AMD or AAA as does measuring blood pressure for diagnosis of hypertension. The specification fails to teach how to identify the normal matrix remodeling process and elastin generated by diseased conditions, and how to interpret the data when the elastin is detected in an individual.

With regard to measuring drusen elastin, *Feeney-Burns et al* (IDS-AX) study age-related changes in the ultrastructure of Bruch's membrane and teach "DEBRIS ON BOTH SIDES OF THE ELASTIC LAMINA WAS THE MOST COMMON FINDING IN OUR SAMPLE. THERE WAS CONSIDERABLE VARIATION IN THE COMPOSITION, AMOUNT, DEGREE OF COMPACTION, THICKNESS, AND EXTENSION OF THE DEBRIS", "IN A GIVEN EYE, DEBRIS IN THE MACULAR SPECIMENS WAS MORE PRONOUNCED THAN THAT IN THE NONMACULAR SPECIMENS". Apparently, increased elastin are present in Bruch's membrane with significant variations in quantity during aging process whether macular degeneration is present or not. The specification fails to teach how to differentiate the aging factor with the risk for AAA.

Therefore, it is evident that at the time of the invention, the skilled practitioner, while acknowledging that the systemic risk factors are involved in macular degeneration, and that measuring the metabolism of ECM components to describe AAA and AMD, still recognize that using such risk factors for diagnosis of AAA was neither routine nor accepted and awaited significant development and guidance for its practice. Therefore, it is incumbent upon applicants to provide sufficient and enabling teachings within the specification for such diagnostic use. Although the instant specification provides reviews, results from a retrospective study, and sketchy ideas for a potential

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use of the claimed methods, it is not enabled to its full scope because the specification fails to disclose any established relationship between elastin levels, and incidence or severity of AAA , and it fails to disclose any particular embodiments reduced to practice to show that either the prevalence or severity of AAA could be predicted according to protein marker changes of AMD, such as elastin and collagen. In summary, the teachings and guidance present in the specification, as a whole, represent an initial investigation into the feasibility of the development of a useful means for AMD diagnosis, which awaits further development to the practical level. Based upon the limited disclosure, the unpredictability of the art, the level of the skill, and the breadth of the claims, one skill in the art would have been required to perform extensive undue experimentation to practice the invention.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-7, 9, 10, 12, 19, 36, 37, and 67 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1-7, 9, 10, 12, 19, and 36 are vague and indefinite because they are incomplete. The method provides for diagnosing an arterial wall disruptive disorder in a subject, however, there is no active and positive step and conclusion in which the method would lead to the diagnosis of such disorder, and which would clearly relate back to the preamble. Method claims need not recite all operating details but should at

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least recite positive, active steps so that the claims will set out and circumscribe a particular area with a reasonable degree of precision and particularity and make clear what subject matter that claims encompass as well as make clear the subject matter from which others would be precluded, *Ex parte Erlich*, 3 USPQ2d 1011 at 6.

Claims 37 and 67 are vague and indefinite because the claim pertains to a kit for performing the immunoassay, there is no description in the specification or claim which would allow one skilled in the art to determine what components are included or excluded in the kit apart from the antibodies.

### ***Claim Rejections - 35 USC § 102***

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 2, 4, 6, 7, 9, 10, 36, 37, and 67 are rejected under 35 U.S.C. 102(a) as being anticipated by *Satta et al* (Eur J Vasc Endovasc Surg 1998 Apr;15:313-9).

Claims are drawn to a method comprising detecting markers for macular degeneration, wherein the marker is elastin, wherein said marker is indicative of arterial wall disorders, preferably AAA. Claims 37 and 67 are drawn to a kit comprising reagents for performing an immunoassay for detection of protein markers, preferably elastin, wherein an antibody specific for a gene product (protein) is used.

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*Satta et al* teach a method comprising detecting elastin in human abdominal aortic aneurysm. An immune detection kit used in the detection of elastin in the sample comprising antibody to elastin (see abstract), and immunoassay reagents for performing the assay. Thus, *Satta et al* anticipate the instant claims.

Please **Note** that the claim recitation "for diagnosis or determining a predisposition to developing an arterial wall disruptive disorder in a subject", has not been given patentable weight because the recitations occur in the preamble. A preamble is generally not accorded any patentable weight where it merely recites the purpose of a process or the intended use of a structure, and where the body of the claim does not depend on the preamble for completeness but, instead, the process steps or structural limitations are able to stand alone. See *In re Kropa v. Robie*, 187 F.2d 150, 152, 88 USPQ 478, 481 (CCPA 1951).

Claims 1, 2, 6, 7, 9, 10, 36, 37, and 67 are rejected under 35 U.S.C. 102(b) as being anticipated by *Juvonen et al* (Eur J Vasc Surg 1994 Jan;8:70-7).

Claims are drawn to a method comprising detecting markers for macular degeneration, wherein the marker is elastin, wherein said marker is indicative of arterial wall disorders. Claims 37 and 67 are drawn to a kit comprising reagents for performing an immunoassay for detection of protein markers, preferably elastin, wherein an antibody specific for a gene product (protein), such as elastin is used.

*Juvonen et al* teach a method comprising detecting elastin in human gastroepiploic artery aneurysm. An immune detection kit used in the detection of elastin in the sample comprising antibody to elastin (see abstract), and immunoassay reagents for performing the assay. Thus, *Juvonen et al* anticipate the instant claims.

***Claim Rejections - 35 USC § 103***

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 1-7, 9, 10, 36, 37, and 67 are rejected under 35 U.S.C. 103(a) as being unpatentable over *Satta et al* or *Juvonen et al* as applied to claims 1, 2, 4, 6, 7, 9, 10, 36, 37, and 67 above, and further in view of *ENCYCLOPAEDIA BRITANNICA*.

Claims are drawn to a method comprising detecting markers for macular degeneration, wherein the marker is elastin, wherein said marker is indicative of arterial wall disorders including various aneurysms. Claims 37 and 67 are drawn to a kit comprising reagents for performing an immunoassay for detection of protein markers, preferably elastin, wherein an antibody specific for a gene product (protein) is used.

*Satta et al* and *Juvonen et al* teach a method comprising detecting elastin in human abdominal aortic aneurysm. An immune detection kit used in the detection of elastin in the sample comprising antibody to elastin (see abstract), and immunoassay reagents for performing the assay. *Satta et al* and *Juvonen et al* do not teach detecting samples from dissecting aneurysm or thoracic aortic aneurysm. However, it is well known in the art as taught by the provision in *ENCYCLOPAEDIA BRITANNICA* that aneurysm could occur in any blood vessel and usually artery resulting from disease of the vessel wall, such as arteriosclerosis. Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to employ the method taught by *Satta et al*

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and *Juvonen et al*, by simply substituting the tissue from AAA or visceral aneurysms with that of TAA or dissecting aneurysm with a reasonable expectation of success.

Thus, the claimed invention as a whole was *prima facie* obvious in the absence of evidence to the contrary.

No claim is allowed.



Any inquiry concerning this communication or earlier communications from the examiner should be directed to Q. Janice Li whose telephone number is 703-308-7942. The examiner can normally be reached on 8:30 am - 5 p.m., Monday through Friday.



If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Karen M Hauda can be reached on 703-305-6608. The fax numbers for the organization where this application or proceeding is assigned are 703-308-8724 for regular communications and 703-308-4242 for After Final communications.

Any inquiry of formal matters can be directed to the patent analyst, Kay Pinkney, whose telephone number is (703) 305-3553.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-1235. The faxing of such papers must conform to the notice published in the Official Gazette 1096 OG 30 (November 15, 1989).

Q. Janice Li  
Examiner  
Art Unit 1632

QJL  
November 8, 2001

  
JAMES KETTER  
PRIMARY EXAMINER  
  
JAMES KETTER  
EXAMINER